

Neonatology

The Newborn

- **The neonatal period** is the 1st 4 weeks of life after birth.
- **Immediate or early neonatal period** is the first week of life.
- **The late neonatal period** is the next 3 weeks of life.
- **The perinatal period** is the period from 20th week of gestational life through the 28th day after birth.
 - As physiological adaptation and adjustment of the baby to the extra uterine environment occur in that period, neonatal mortality accounts for 65 % of deaths in the 1st year of life and it is highest during the 1st day after birth.

Classifications

According to:

A. Gestational period:

1. Preterm less than 37 wks of gestation
2. Full term between 37 and 42 wks of gestation
3. Post term more than 42 wks of gestation.

B. Birth weight:

1. Large birth weight (macrosomia) = 4000 gm or more after birth.
2. Normal birth weight (NBW) = 2500 to 4000 gm after birth.
3. Low birth weight (LBW) = 2500 gm or less after birth.
4. Very low birth weight (VLBW) = 1500 gm or less after birth
5. Extremely low birth weight (ELBW) = 1000 gm or less after birth
6. Impossibly low birth weight (ILBW) = 750 gm or less after birth

C. Size for gestation:

1. Small for gestational age (SGA)" small for date " light for date":
Birth weight less than 10th percentile for gestational age.
2. Appropriate for gestational age (AGA):
Birth weight between 10th and 90th percentile for gestational age.
3. Large for gestational age (LGA):
Birth weight more than 90th percentile for gestational age.



Postnatal differentiation between PT & FT:

	Preterm	Full term
1. Measurements		
Weight	< 2.5 kg	3-3.5 kg
Length	< 47 cm	50 cm
Head circumference	< 33 cm	35 cm
Chest circumference	< 30 cm	33 cm
2. Vitality		
Cry	Feeble	Good
Sucking power	Weak	Good
Muscle tone	Poor	Good
Muscle movement	Slow, infrequent	Strong, frequent

Characteristics of the normal FT newborn

1. Body proportions :

- * The head : It is relatively large, the face is rounded & the mandible is relatively small.
- * The chest : It is rounded rather than flattened antero-posteriorly.
- * The abdomen : It is prominent.
- * The extremities are relatively short.
- * The mid-point of the stature is at the umbilicus (in adult at the symphysis pubis).
- * The predominant posture is partial flexion.

2. Color :

- * The normal color is rosy pink.
- * Mongolian spots : It is purplish mottling over the back & buttocks (not pathological).

3. Skin :

- * At birth the infant is covered with cheesy white material adherent to the skin called vernix caseosa.
- * The normal skin is dry, peeling is found at the ankles but extensive peeling & transverse fissuring over the abdomen denotes dysmaturity.
- * At birth lanugo hair usually covers shoulders.

* Milia : It is distended sebaceous glands appears as white spots over nose & cheeks (needs no ttt).

* Capillary hemangiomas are common over the eye lids , forehead & back of the neck .

* Umbilical stump darkens in color , dries & then falls by the end of the first week.

4. Measurements :

* The length : 50 cm.

* The weight : 3.400 kg.

* Head circumference : 33 cm.

* RR : 30-40 / min.

* Pulse : 120-160 / min .

* BP : 70-85 / 40-60 mmHg.

* Temperature : At delivery the infant's temperature is the same as his mother . After delivery there is a transient fall in temperature which is usually restored within 4-8 hours.

5. cardiac signs :

* The heart sounds have the characteristic tic tac rhythm .

* There may be transient innocent cardiac murmurs .

* CXR : the heart seems to be large in respect to the shape of the chest so the apex is located in the 4th intercostal space outside the mid-clavicular line.

6. G.I.T. :

* The 1st stools will be generally passed within 24 hrs which consist of meconium .

* By establishing breast feeding , meconium stools start to be replaced by transitional stools on the 3rd or 4th day (greenish brown) , later become golden yellow.

* Frequency : 3-5 / day by the end of the 1st week .

* Melena in the newborn may be due to :

a. Swallowed blood \$.

b. Hemorrhagic diseases of the newborn .

7. Renal functions :

* Urination may be delayed for 24-72 hrs , depending on external atmospheric temp.

* The kidney is not able to concentrate urine.

* Urine may contain abundant urates which give the pink stain of urine.

* GFR is also present & may lead to retention of drugs and toxicity.

8. Genitalia & mammary glands :

- * Enlargement & secretions of the breasts in both sexes may occur.
- * Hypertrophy & enlargement of both labia may occur in females.
- * Both conditions are due to passage of maternal estrogen through the placenta.

9. Blood :

* Hb % : 17-19 gm % with mild reticulocytosis & normoblastemia.

* WBCs :

- After the 1st 24hrs : 25000 / cmm with relative neutrophilia.
- After one week : 15000 / cmm with relative lymphocytosis.

* Coagulation factors : There is little transfer of certain coagulation factors from the mother. Establishment of normal hemostatic mechanisms depends on early development of normal bacterial flora & elaboration of adequate amount of vit. K.

The post-term newborn

Definition

Baby delivered after 42 weeks of gestation regardless of birth weight.

Etiology

1. Idiopathic.
2. Chromosomal abnormalities.
3. Neural tube defects, because an intact feto pituitary adrenal axis is involved in initiation of labor.

Physical characteristics

1. He is alert, with open eyes.
2. The skin is pale desquamated, wrinkled & dry.
3. Absent lanugo hairs.
4. Decreased or absent vernix caseosa.
5. Long nails.
6. ± Meconium staining.

Problems of post-maturity

The mortality rate is 3 times that of the term infants due to :

1. Congenital anomalies which may be the cause of post-maturity.

2. Meconium aspiration.
3. Persistent pulmonary hypertension.
4. Hypoglycemia, hypocalcemia, polycythemia.
5. Perinatal depression.

Management of the post-mature neonate.

Delivery of a post-term newborn should take place in an equipped hospital with a neonatal intensive care unit.

1. Routine delivery room & nursery care plus :
 - a. Resuscitation if needed.
 - b. Ante-, intra-, & post-partum monitoring of the fetal well being.
2. Management of the above mentioned complications

The large for gestational age newborn

Definition

Baby whose birth weight measures $> 90^{\text{th}}$ percentile (or $> 2 \text{ SD}$) for his calculated gestational age.

The LGA could be FT, PT, post-term.

Etiology

1. Constitutional (large parents).
2. Infant of diabetic mother.
3. All causes of hydrops fetalis.
4. Beckwith – Wiedemann \$.

Problems of LGA

1. Special problems related to the underlying cause .
2. Transient tachypnea of the newborn especially if born by C.S.
3. Birth injuries.
4. Hypoglycemia.(IDM, BW\$, Erythroblastosis fetalis).

Management of LGA

Delivery of a LGA newborn should take place in an equipped hospital with a neonatal intensive care unit.

1. Routine delivery room & nursery care plus :
 - a. Resuscitation if needed.
 - b. Ante-, intra-, & post-partum monitoring of the fetal well being.
2. Management of the above mentioned complications

Apgar Score

Measuring the score at $\Rightarrow$

1. 1 minute $\Rightarrow$ as indicator for the presence of asphyxia & helps to decide the needed mode of resuscitation.
2. 5 minutes $\Rightarrow$ is a more accurate indicator for later neurologic residual damage.

The following signs are observed & given a score 0, 1 or 2.

Score \ Sign	0	1	2
HR	Absent	< 100	> 100
Respiration.	Absent	Slow & irregular	Good crying
Muscle tone	Absent	Some flexion of extremities	Active movements
Reflex to nasal catheter	Absent	Grimace	Cough, sneeze
Color	Blue or pale	Body pink & extremities blue	Completely pink

A. A score of 8-10

$\Rightarrow$ Indicates good general condition & no asphyxia.

1. Gently, suction air way including nares.
2. Good dryness of the baby including the head.
3. Maintain body temperature.

B. A score of 5-7:

$\Rightarrow$ Indicates mild asphyxia

1. Stimulate breathing by repeated slapping of the soles, or rubbing of spine or sternum.
2. Provide O₂ by bag & mask.
3. If the mother has received narcotics during labor $\Rightarrow$ Give O.01mg / Kg IM

↓

If improving

↓

If deteriorating

↓

Continue as A

↓

Continue as C

C. A score of 3-4

$\Rightarrow$ Indicates moderate asphyxia.

1. Ventilate with bag & mask, using 100% O₂, till improvement & spontaneous respiration begins.
2. If the heart rate is less than 60 $\Rightarrow$ intubate the baby & cardiac massage at a rate of 120/min is done.

D. A score of 0-2:

⇒ Indicates severe asphyxia.

1. Proceed directly to intubation & bag ventilation with 100% O₂
2. Perform cardiac massage.
3. If no improvement within 2 minutes ⇒ insert umbilical catheter & give the following drugs.
 - a. NaHCO₃ ⇒ to correct metabolic acidosis.
 - b. Glucose 10-25% ⇒ to correct hypoglycemia.
 - c. Epinephrine 0.01mg/Kg IV or endotracheal & may be repeated every 5 minutes.
 - d. Finally ⇒ Ca-gluconate 1-2 ml/kg slowly IV.
- All these drugs are ineffective in absence of adequate O₂-ventilation.



Come on .. Let's gooooo

Neonatal Reflexes

Moro reflex

Stimulus

1. Making a loud noise near the ear.
 2. Sudden withdrawal of the blankets from underneath the infant.
 3. Sudden dropping of the head for about 15 in the examiners hand.
 4. Putting a cold hand or stethoscope on the baby's skin.
- It is produced by excitation of cervical or labyrinthine proprioceptors by movements.*

Response

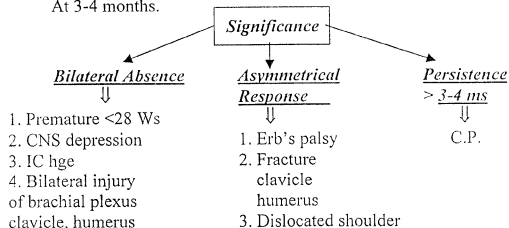
Extension of the trunk + Extension & abduction then flexion & adduction of the arms.

Age of appearance:

Birth (28 weeks)

Disappears.

At 3-4 months.



Placing Reflex

If the dorsum of foot touches the under edge of the table.

⇒ The foot is lifted and placed on the table.

Appearance : At birth.

Disappears : At 1 ½ months.

Absence : CNS depression or LL paresis.

Stepping reflex

If soles of feet touch the surface of the table in the erect position ⇒

Inclining the body forwards ⇒ Regular alternating steps forwards.

Appearance : At birth.

Disappears : At 1 ½ months.

Absence : CNS depression or LL paresis.

Rooting & suckling Reflexes

Stimulation of the cheek $\Rightarrow$ turning of the face & mouth towards the stimulus then stroking the lips $\Rightarrow$ Repeated suckling movements by the mouth.

Appearance : At birth.

Disappears : At 4 ms awake & 7 ms asleep.

Absence : CNS depression.

PT.

Bulbar palsy.

Grasp Reflex

Light touch to the palm of the hand $\Rightarrow$ the baby grasps

Appearance : At birth.

Disappears : Palmar at 6 ms & planter at 10 ms.

Absence : CNS depression .

Brachial plexus injury

Persistence : C.P.

Glabellar reflexes

Sharp tap on the glabella $\Rightarrow$ momentary tight closure of the eyes.

Appearance : 32-34 weeks.

Disappears : Variable.

Absence : CNS depression.

PT.

Pupillary Reflex

Close one eye & expose the other to light $\Rightarrow$ pupillary constriction.

Appearance : 29-37 weeks.

Disappears : Persists.

Absence : CNS depression.

PT.

Impaired vision.

Tonic neck reflex

Put the baby in supine position $\Rightarrow$ turn the head rapidly to one side $\Rightarrow$ Extension of limbs on the side to which the face is turned & flexion of limbs on the other side.

Appearance : At 2 ms.

Disappears : At 6-7 ms.

Absence : Medullary or spinal cord lesions.

Neck righting reflex

Put the baby in supine position $\Rightarrow$ turn the head slowly to one side $\Rightarrow$ Rotation of the trunk to the direction of the head.

Appearance : 32-34 weeks.

Disappears : 24 ms.

Absence : It is absent or decreased in infants with spasticity

Parachute reflex

If the infant is suspended in the prone position & suddenly allowed to fall for a short distance $\Rightarrow$ Extension of arms, hands & fingers occur. Appears at 9 ms & persists.

It is absent or decreased in infants with spasticity.

Landau reflex

If the infant is suspended in the prone position with the examiner's hands below the abdomen $\Rightarrow$ Extension of the head, trunk & hips. then flex the infant's head $\Rightarrow$ Flexion of the trunk & hips

Appears at 3 ms & disappears at 24 ms.

It is absent or decreased in infants with spasticity.

Other reflexes.

1. *Deep tendon reflexes* $\Rightarrow$ normally present at birth usually brisk.
2. *Babinski sign* $\Rightarrow$ An extensor response is usually obtained during the 1st year of life.



Birth Injuries

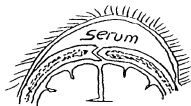
Head injuries

Caput Succedaneum

- It is diffuse edematous swelling of the soft tissues of the scalp involving the presenting part of the fetus at birth.
- It is maximum at birth & disappears gradually within few days.
- It crosses suture lines
- No t t t is needed.

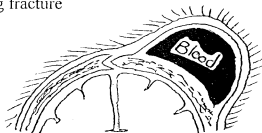
Cephalhematoma.

- It is subperiosteal hge of the skull
- Characters
 1. It doesn't cross suture lines.
 2. It is not present at birth, then gradually develops within hours.
 3. No discoloration of the overlying skin as in caput succedaneum.
 4. In 25% $\Rightarrow$ there's an underlying fracture



Complications.

1. Anemia.
2. Hyper-bilirubinemia.
3. Infection.
4. Disfigurement of the skull
5. Organization with fibrosis & calcification.



Treatment

1. Depressed fracture $\Rightarrow$ Correction.
2. Shock or anemia $\Rightarrow$ Blood or packed RBCs transfusion.
3. Hyperbilirubinemia $\Rightarrow$ phototherapy or exchange transfusion.
4. Infection $\Rightarrow$ antibiotics & drainage of pus if formed.

Intracranial Hemorrhage.

Etiology

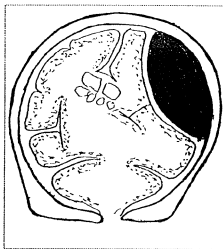
1. Trauma $\Rightarrow$ Forceps.
2. Anoxia.
3. Hgic blood disease.
4. Vascular anomalies.

5. Prematurity: in the PT the blood vessels lining the ventricles are fragile & have little support & also lack the normal autoregulation which copes with changes in the BP to maintain the cerebral perfusion.

C/P

It may present at birth or later on

1. Poor feeding.
2. Disturbed level of consciousness which may reach to coma.
3. Convulsions.
4. Absent Moro reflex.
5. Central RD $\Rightarrow$ slow irregular respiration apnea & cyanosis.
6. Manifestations of $\uparrow$ ICP
 - Forceful vomiting.
 - High pitched cry
 - Convulsions & tense bulging fontanel
7. Eye signs $\Rightarrow$ unequal pupils
Failure to react to light.
Nystagmus
Retinal hge.



Investigations

1. Brain Sonar.
2. Brain CT-scan.
3. MRI

Treatment

A. Prevention.

1. Good obstetric care.
2. Prevention of prematurity.

B. Curative.

1. Incubator care.
 - a. Position of the newborn. $\Rightarrow$ Raise the head 30 degrees $\rightarrow$ why?
 - b. Tube feeding or IV-Fluids.
2. Convulsions.
 - $\Rightarrow$ Phenobarbitone.
3. Hgic blood diseases.
 - a. Vit K1 $\Rightarrow$ 1-5 mg IM
 - b. FFP or fresh blood

- c. 4. Surgical evacuation or tap. & if hydrocephalus develops
⇒ shunt operation.

Peripheral Nerve Injury

Erb's Paralysis

Etiology

Injury of C5 & C6

Position

The affected UL is:

1. Adducted & internally rotated at the shoulder
2. Extended at the elbow
3. Pronated forearm
4. Flexed at wrist.

Policeman's tip position

5. The paralyzed limb of LMNL i.e. hypotonia

Loss of biceps reflex "C5 & 6"

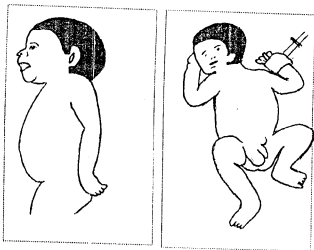
Atrophied biceps.

Unilateral absent Moro-reflex.

6. The hand moves well

i.e. intact grasp reflex.

7. Sensory impairment on the outer aspect of the arm.



ttt

⇒ "Airplane- splint" ⇒ the arm is abducted 90 degree + external rotation at the shoulder + full supinated forearm + slight extension at the wrist for 1 week then start physiotherapy.

Klumpke's paralysis

Etiology

Injury of C7, C8 & T1

C/P

1. Paralysis of the muscles of the hand ⇒ loss of hand grip & grasp reflex.
2. Horner syndrome ⇒ is usually associated due to interruption of cervical sympathetic fibers in T-1
⇒ Ptosis, miosis, anhydrosis
& enophthalmos on the same side.

!!!

Intermittent padding of the fist with small ball & splinting the wrist in neutral position for 1 w then start physiotherapy

N.B.

If paralysis persist for 2-5 months neuroplasty is indicated.

Phrenic nerve paralysis

Etiology:

Injury of C 3,4 & 5.

C/P:

1. cyanosis & irregular respiration.
2. Breathing is thoracic in type > no bulging of the abdomen with inspiration.
3. Diminished air entry on the affected side.

Investigations:

Fluoroscopy : elevation of diaphragm on the paralyzed side during inspiration.

!!!

1. The infant should be placed on the paralyzed side.
2. O₂ is given if necessary.
3. Progressive naso-gastric tube feeding or IV fluids if severe RD.
4. Surgical plication is rarely indicated.

Facial Nerve Palsy

⇒ due to compression of the facial nerve by forceps delivery.

C/P

There is deviation of the mouth to healthy side, failure to close eye, absent naso-labial fold & feeding troubles due to buccinator paralysis.

!!!

1. Care of the eye & feeding.
2. Neurplasty ⇒ if paralysis > 3ms

Sternomastoid muscle injury

- ⇒ due to neck traction during delivery ⇒ hematoma of the sternomastoid ⇒ fibrosis ⇒ calcification ⇒ firm mass "Sternomastoid mass".
⇒ shortening of the ms ⇒ torticollis of the neck.

!!!

1. Physiotherapy.
2. Surgical release of the affected ms in case of failure of physiotherapy.

Visceral Injury

- ⇒ Due to rough manipulation during breech delivery.
- Subcapsular hematoma of the liver, rupture spleen & adrenal hge are the commonest.
- ⇒ severe pallor, jaundice, shock & palpable abdominal ms.

Low Birth Weight

Definitions

1. Low Birth weight "LBW"
Wt < 2.5 Kg at birth
2. Very Low Birth weight "VLBW"
Wt < 1.5 Kg at birth.
3. Preterm
Any live-born baby delivered before 37 weeks.
4. Small - For date
The birth weight is less than the 10th percentile for his calculated gestational age. "due to IUGR"
5. Post-term
Infant born after 42 weeks

Etiology

A. Preterm de4livery About 60% of cases.

1. Idiopathic : In 50% of cases.
2. Twin pregnancy.
3. Ante-partum Hge.
4. Toxemia.

5. Diabetic mother.
6. Premature separation of the placenta.
7. Badly timed C.S.

B. IUGR "Small-for-date" About 40% of cases.

1. Ante-natal infections: STORCH.
2. Congenital anomalies of the fetus.
3. Chromosomal aberrations.
4. Placental dysfunction syndrome
⇒ Fetal malnutrition.

How to differentiate between premature & IUGR?

⇒ By assessment & calculation of gestational age.

Assessment of gestational age

History

1. Date of LMP.
2. Date of onset of fetal movement "18-20 Ws"

Ante-natal examination.

Fundal level.

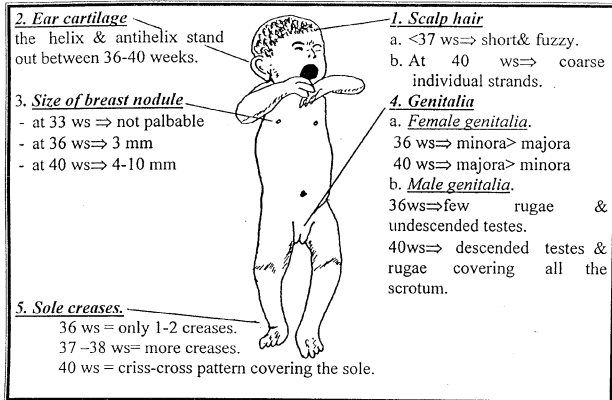
Ante-natal investigations.

1. U/S ⇒ Bi-parietal diameter.
Bi-acromial diameter.
2. Amniotic fluid analysis.
For lung ⇒ L/S ratio.
For kidney ⇒ Creatinine.

You can do it ...

At Birth

A. Physical Signs.



B. Neurologic evaluation.

1. Pupillary light reflex $\Rightarrow$ 29-31 ws.
2. Moro reflex $\Rightarrow$ 28-31 ws
3. Glabellar reflex $\Rightarrow$ 32-34 ws.
4. Rooting reflex $\Rightarrow$ 31-33 ws.
5. Suckling reflex $\Rightarrow$ 31-33 ws.

The Preterm Baby

Criteria of Prematurity

1. Birth wt $\Rightarrow < 2.5$ Kg.
2. Length $\Rightarrow < 47$ Cm.
3. Skull circumference $\Rightarrow < 33$ Cm.
4. Chest circumference $\Rightarrow < 30$ Cm.
5. Foot length $\Rightarrow < 7$ Cm.
6. Absent ossific center in the cuboid bone.

Handicaps & Complication of prematurity

Respiratory.

1. Immature respiratory center $\Rightarrow$ irregular, periodic respiration & recurrent apnea.
2. $\downarrow$ Surfactant $\Rightarrow$ RDS.
3. Weak respiratory ms & weak thoracic cage.
4. Weak cough & gag reflexes $\Rightarrow$ aspirations.
5. Poor function of the cilia $\Rightarrow$ chest infection.
6. Immature pulmonary capillaries $\Rightarrow$ pulmonary hge.

Inadequate regulation of body temp.

1. Immature heat regulating system.
2. $\uparrow$ Surface area in relation to body wt.
3. $\downarrow$ S.C. Fat $\Rightarrow$ $\uparrow$ heat loss.
4. $\downarrow$ Ms. activity $\Rightarrow$ $\downarrow$ heat production.

Renal

1. $\downarrow$ GFR
2. Inability to concentrate urine.

Hepatic

1. Delayed activation of hepatic enzymes $\Rightarrow$ physiological jaundice.
2. $\downarrow$ storage capacity of the liver esp. for glycogen $\Rightarrow$ $\uparrow$ liability for hypoglycemia.

Hemostatic handicaps.

1. $\downarrow\downarrow$ clotting factors $\Rightarrow$ due to hepatic immaturity.
2. $\uparrow\uparrow$ capillary fragility $\Rightarrow$ due to inadequate supporting tissues.

CVS.

1. $\uparrow\uparrow$ shunting of blood through PDA & foramen ovale in response to hypoxia, stress & polycythemia $\Rightarrow$ circulatory insufficiency & under perfusion of vital organs.
2. Persistent P++ $\Rightarrow$ $\uparrow\uparrow$ blood shunting.

Immunologic handicaps.

1. Defective transfer of Igs "IgG".

CIT

2. Immature immune functions as phagocytosis, T-cell function, B-cell function & $\downarrow\downarrow$ complement .So sepsis is more common.

1. Weak suckling & swallowing reflexes.
2. $\downarrow$ capacity of stomach & low gastric acidity.
3. $\downarrow\downarrow$ bile salts $\Rightarrow$ fat & fat soluble vitamins malabsorption.
4. Diminished body stores of minerals. & vitamins e.g. Iron & VitD.
5. Delayed neuro-muscular coordination. $\Rightarrow$ freq. vomiting.

Complications of prematurity.

1. Respiratory.

- Hyaline Membrane Disease.
- Atelectasis $\Rightarrow$ either 1ry or 2ry.
- Pulmonary hge.
- Recurrent apnea.

2. Hyperbilirubinemia & kernicterus.

3. IC- hge
4. Intercurrent infections $\Rightarrow$ E-coli & staph.
5. Retro-lental fibroplasia.
 $\Rightarrow$ due to prolonged use of O₂ at high tension .
6. Hypothermia.
7. Hypoglycemia.
8. NEC.
9. Persistent PDA.
10. High incidence of organic brain damage.
11. High incidence of rickets & iron-deficiency anemia.



*It's your own way ..
prove yourself*

Small-for date "Dysmature baby"

Physical character.

1. Muscle wasting due to intra-uterine malnutrition.
2. Skin $\Rightarrow$ thin loose, dry & cracked, with minimal S.C. fat.
3. Scalp hair $\Rightarrow$ sparse & may fall completely.
4. Widely opened fontanels & sutures.
5. The umbilical cord is very thin & may be meconium stained.

Complications

1. Perinatal asphyxia.
2. Hypoglycemia.
3. Hypothermia.
4. Pulmonary hge.
5. Meconium aspiration.
6. NEC.
7. Polycythemia.
8. Illness related to cong. anomalies or cong. infections.



We will always be with you

Management of LBW

Prevention.

Good ante-natal care is essential to avoid the preventable causes as:

1. Ante-natal STORCH infections.
2. Control of maternal diseases e.g. DM, hypertension etc.
3. Ante-natal care of maternal nutrition.
4. Avoid exposure to teratogens & irradiations.
5. Avoid unnecessary C.S.

Active t t t

I. Incubator care.

1. For regulation of body temp.
2. Regulation of relative humidity "40-60%.
3. Regulation of O₂ concentration.
4. Reduction of the possibility of contamination.

II. Feeding.

Onset → as early as possible for fear of hypoglycemia.

Mode of feeding.

1. If he can suckle & swallow ⇒ breast or bottle.
2. If he can swallow only ⇒ breast milk by dropper or spoon.
3. If he can't suckle or swallow ⇒ nasogastric tube feeding.
4. If there is respiratory distress, IC-hge, NEC & Other causes of intestinal obstruction ⇒ I.V. fluid therapy.

Type of feed.

1. Breast milk of the preterm baby is more suitable than full term breast milk ⇒ why?
2. Humanized milks are suitable for most infants.
3. Special preterm formula is available.

Amount of feed.

We start 5-10 ml / fed & ↑ gradually according to tolerance of feeding.

N.B.

Regurgitation, vomiting, abdominal distension or residual from the previous feed upon gastric suction ⇒ suspicion of sepsis or NEC should be considered.

Fluid Requirements.

1. E.T. ⇒ we start by 60 ml / kg in day "1" ⇒ ↑ 10-20 ml / kg / D till max. 150 ml / kg / D
2. P.T ⇒ 80ml / kg in day "1" ⇒ ↑ 10-20 ml/Kg / D till max 150 ml / kg / D.

III. Prevention of infections.

1. Every thing should be completely sterile.
2. Prophylactic antibiotics.
Penicillin + Garamycin.

Immunoglobulin preparations may be given.

IV. Prevention of nutritional disorders

1. Vit - K1 $\Rightarrow$
1-5 mg IM immediately after birth.
2. Vit-E $\Rightarrow$ to prevent hemolysis.
3. Vit - D, C & Iron should be started as early as 2nd month.



Happy Mother's Day

Increase your efforts ...

Neonatal Respiratory Distress

It may be due to

<i>Parameter</i>	<i>CNS-failure</i>	<i>Peripheral resp. diff</i>
Causes	1. Maternal narcosis 2. Perinatal anoxia 3. IC he. 4. CNS anomalies 5. Hypothermia 6. Hypoglycemia 7. Septicemia	<u>A. Pulmonary causes</u> 1. Idiopathic RDS 2. Iry atelectasis. 3. Aspiration of amniotic fluid. 4. Penumonia⇒ cong. Or acquired. 5. Pneumothorax. 6. Lobar emphysema. <u>B. Extra-pulmonary causes</u> 1. Choanal atresia. 2. Diaphragmatic hernia. 3. Tracheo-esophageal fistula. 4. Focal cord paralysis. 5. Laryngeomalacia.
C/P	1. Apnea 2. Slow, irrregular, gasping respiration. 3. Cyanosis.	1. Tachypnea. 2. Working alae nasi 3. Expiratory grunting 4. Intercostal & subcost. retractions. 5. Chin tug. 6. Cyanosis.

Idiopathic Respiratory Distress Syndrome Hyaline Membrane Disease

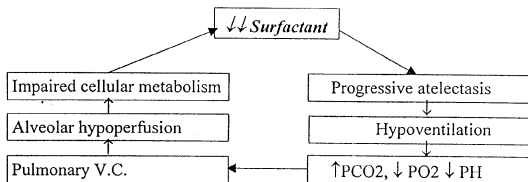
It is a major cause of neonatal death esp. in PT

Predisposing Factors

1. Prematurity.
2. Maternal DM.
3. C.S.
4. Perinatal anoxia.
5. Hypotention, hypovolemia & cold stress.

Etiology & Pathogenesis

- It is due to absence or deficiency of surfactant which is a phospholipid secreted by type II alveolar cells to line the alveoli.
- Surfactant $\Rightarrow$ $\downarrow\downarrow$ surface tension of the alveolar walls $\Rightarrow$ prevent alveolar collapse during expiration.
- In hyaline membrane disease "HMD": the following sequence occurs.



C/P

1. Tachypnea.
2. Working alae nasi
3. Expiratory grunting
4. Intercostal & subcostal retractions.
5. Chin tug.
6. Cyanosis.
7. Diminished air entry, fine & medium sized consonating crepitations on chest auscultation.

Investigations

1. CXR : reticulo-granular appearance.
2. Blood gases .
3. Shake test.

ttt

A. Prevention.

1. Avoidance or prevention of predisposing factors e.g.
 - a. Prematurity through appropriate management of high risk pregnancy & labor.
 - b. Avoidance of unnecessary C.S.

c. Careful management of diabetic mother.

2. I.U. Diagnosis & treatment.

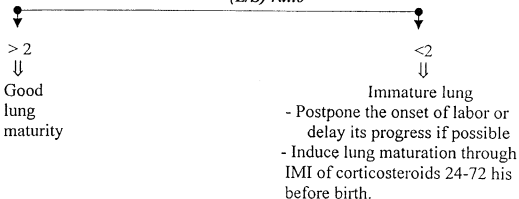
a. Calculation of gestational age through $\Rightarrow$ Date of LMP

U/S $\Rightarrow$ Skull diameter.

Bi-acromial diameter.

b. Amniotic fluid sample & measure lecithin / sphingomyelin

(L/S) ratio



3. Ante-natal & intrapartum monitoring of the baby to reduce the risk of fetal asphyxia.

4. Prevention of cold stress & birth asphyxia $\Rightarrow$ $\downarrow$ Risk of RDS.

B. Curative

- The ttt aims at breaking the vicious cycle mentioned before & supporting the newborn until he restarts to synthesize his own surfactant.
- Incubator care is usually necessary for prevention of hypothermia, O₂ administration, humidity & providing adequate IV fluids & nutrition are important.

1. Correction of hypoxia.

- Warm humidified O₂.
- Follow up by arterial blood gases.
- PO₂ should be kept at 60-80 mmHg
- If 70-100%O₂ is not sufficient to correct hypoxia $\Rightarrow$ Mechanical ventilator is indicated.
- Indications of assisted ventilation are :
 - a. pH < 7.2.
 - b. PCO₂ > 60 mmHg.
 - c. PO₂ < 50 mmHg at O₂ concentration of 70-100 %.
 - d. Persistent apnea.
- Q: Mention O₂ - toxicity ?

2. Correction of metabolic acidosis.

NaHCO₃ slowly IVI according to blood gases.

3. Antibiotics

- If infection occurs the appropriate antibiotic should be used.

4. Synthetic surfactant.

It is used as slow endo-tracheal administration through a special apparatus connected to the endotracheal tube.

5. Exchange transfusion.

May be used in ttt of RDS

⇒ Why?

6. General supportive care.

- Adequate nutrition.
- Prevention of hypothermia.

Complications of HMD & intensive care management

1. CNS : a. Central hypoxia.
b. Encephalopathy.
c. Intraventricular hge.
2. Complications of endotracheal intubation & ventilation :
a. Pneumothorax.
b. Pneumopericardium.
3. Complications of umbilical vessels catheterization :
a. Sepsis.
b. NEC.
c. Portal vein thrombosis.
4. O₂ toxicity :
a. Retinopathy.
b. Bronchopulmonary dysplasia.
5. Anemia due to frequent blood samples

Prognosis

Depends on early diagnosis and management in well equipped NICU by skilled personnel.

In good centers, about 50% of those under 1 kg survive and the morbidity progressively decreases at higher weights to over 95% in sick infants weighing more than 2.5 kg.

The long term prognosis for normal pulmonary function in most infants surviving HMD is excellent.

Transient tachypnea of the newborn RDS type II

Etiology

It is caused by delayed or slow absorption of fetal lung fluid.
- It is common in PT or FT who delivered by C.S.

C/P

1. Tachypnea of early onset may be the only finding.
2. Other signs of RD including grunting & cyanosis which are relieved by minimal O₂.
3. Recovery is usually rapid within 3 days.
4. Auscultation may show no crepitation or wheezes

Investigations.

CXR⇒ Prominent vasculature.
Fluid lines in lung fissures.

ttt

1. O₂ therapy
2. Stop oral feeding till tachypnea improves.



*We should help each other ..
It's one community*

Meconium aspiration syndrome

Risk factors:

1. Perinatal asphyxia.
2. Breech delivery.

C/Ps:

1. Tachypnea.
2. Working alae nasi
3. Expiratory grunting
4. Intercostal & subcostal retractions.
5. Chin tug.
6. Cyanosis.

Investigations:

CXR $\Rightarrow$ patches of lung collapse & over inflation.

ttt

1. Proper clearing of the airway by gentle suction may be life saving.
2. Gentle supportive therapy with minimal interference probably achieves the best results.
3. Supportive ventilation may aggravate the lung disorders.

Neonatal Apnea

Definition

It is cessation of breathing for more than 20 seconds or long enough to produce cyanosis & bradycardia

Etiology

1. Infections $\Rightarrow$ Sepsis.
Meningitis.
NEC
2. CNS $\Rightarrow$ Hge.
Kernicterus
Immaturity of respiratory center.
3. Respiratory $\Rightarrow$ RDS
Severe pneumonia.
Pneumothorax.
Atelectasis.
4. Metabolic $\Rightarrow$ Hypoglycemia.

Hypocalcemia.
Hypoxia.
Hypothermia.
Hypo & hypernatremia

5. GIT $\Rightarrow$ Gastro-esophageal reflux
NEC

ttt

1. Identification & ttt of the cause.
2. Stimulation of respiration by
 - a. Tactile stimulation of the skin.
 - b. Mask & bag may be needed.
 - c. Theophylline : IVI.
Loading $\Rightarrow$ 5mg/Kg
Maint $\Rightarrow$ 24 mg/Kg/D
3. Nasal CPAP $\Rightarrow$ may be needed if the previous measures fail.
4. Mechanical ventilation may be needed.

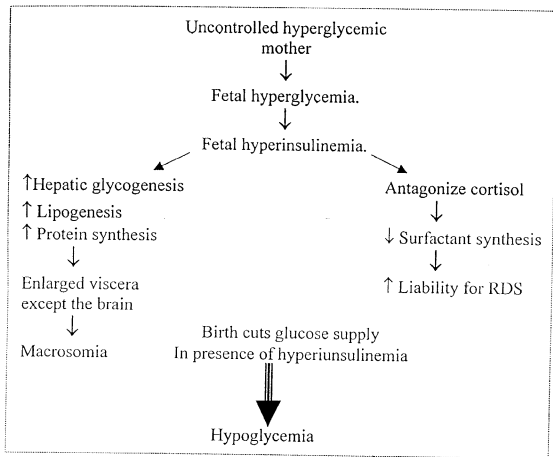


Make success your destiny

Infant of Diabetic Mother

It must be correctly called "Infant of a non controlled hyperglycemic diabetic mother".

Patho-Physiology



C/P

1. Large & plumpy $\Rightarrow$ due to enlarged viscera.
2. Plethoric face $\Rightarrow$ due to polycythemia.
3. Jitteriness or tremors, eye rolling even true convulsions.
4. Intermittent apnea spells or tachypnea.
5. Episodes of cyanosis.
6. Poor feeding.

*It's hard to win ...! but
it's better with this life*

N.B.

There is a high incidence of certain complication in addition to hypoglycemia

As:

1. RDS
2. Polycythemia.
3. Neonatal jaundice.
4. Hypocalcemia & hypomagnesemia.
5. Congenital anomalies.
6. HF
7. Severe hypoglycemia $\Rightarrow$ organic brain damage $\Rightarrow$ MR
8. Birth trauma $\Rightarrow$ due to macrosomia.

ttt

A. Before birth.

Careful efficient ante-natal care & good control of maternal diabetes.

B. After delivery.

1. Measurement of blood glucose every hour in the 1st 8hrs.
2. If clinically well with no hypoglycemia $\Rightarrow$ give early oral feeding every 2hrs.
3. If blood glucose < 35 mg/dl $\Rightarrow$ give IV infusion of glucose even if no symptoms.
4. Hypocalcemia $\Rightarrow$ 2-4 ml/kg calcium gluconate slow IV.
5. Hypomagnesemia $\Rightarrow$ Mg SO₄ 50% ml/kg.
6. ttt of other complications if present.



Be with us .. It's success

Neonatal Hypoglycemia

Definition

Neonatal hypoglycemia is defined as blood glucose

* < 35 mg/dl during the first 3 hrs.

* < 40 mg/dl between 3-24 hrs.

* < 45 mg/dl after 24 hrs.

Etiology

A. Inadequate substrate or enzyme function :

1. Intra-uterine growth retardation.
2. Prematurity
3. Infant of toxemic mother.
4. Infant with placental abnormalities.

B. Hyperinsulinemia :

1. Infant of diabetic mother. At fetal hyperglycemia
2. Rh-incompatibility.
3. Islet cell hyperplasia.
4. Beckwith Wiedmann S.
5. Panhypopituitarism.

C. Increased metabolic needs:

1. RDS.
2. Perinatal asphyxia.
3. Polycythemia.
4. Hypothermia.
5. Systemic infections.
6. Congenital cyanotic heart diseases.
7. Infants with heart failure.

D. Inborn errors of metabolism:

1. Galactosemia.
2. Glycogen storage diseases.
3. Methyl malonic acidemia.

C/P

⇒ As infant of diabetic mother.

ttt

1. High risk infant with normal blood glucose ⇒ early oral feeding/2-3 hrs.
2. If oral feeding is not tolerated ⇒ give IV infusion of glucose.

3. Hydrocortisone 10 mg / kg / D
or prednisone 1mg / kg / D
⇒ may be used in persistent hypoglycemia.
4. Glucagon ⇒ has been used with success.
5. Monitoring blood glucose / 2 hrs then every 4-6 hrs when the blood glucose became controlled.
6. tt of the etiology.



Ok .. Do it ...

Neonatal Jaundice

Definition

It is yellowish discoloration of skin, conjunctiva & mucus membranes due to increased serum bilirubin above 7mg /dl.

Bilirubin Metabolism

See chronic hemolytic anemia

Etiology

A. 1st. day

1. Hemolytic diseases of the newborn until proved otherwise.
 - Rh incompatibility
 - ABO & other groups incompatibility
2. Ante-natal infections i.e. TORCH-EB

B. 2nd -3rd day.

1. Physiological jaundice.
2. Crigler-Najjar syndrome "familial non-hemolytic jaundice"
3. Ante-natal infections.

C. 3rd -7th day.

1. Neonatal sepsis.
2. Absorption of hematoma e.g.
 - Cephalhematoma, IC hge.
 - Subcapsular hematoma of the liver.
3. Hereditary spherocytosis & G6PDD
4. Ante-natal infections.

D. After the 1st week

1. Congenital biliary atresia.
2. Neonatal hepatitis.
3. Septicemia & infections.
4. Hereditary spherocytosis & elliptocytosis.
5. G-6-PD deficiency "after intake of oxidizing agent.
6. Breast milk jaundice.
7. Galactosemia.
8. Inspissated bile syndrome.
9. Synthetic vit. K injection
10. Choledocal cyst.

E. Persistent after the 1st month.

1. Congenital biliary atresia.
2. Neonatal hepatitis.
3. Hypothyroidism.
4. Intestinal obstruction.

5. Congenital infections.
6. Galactosemia.
7. Inspissated bile syndrome.

According to the type of hyperbilirubinemia

I. Unconjugated hyperbilirubinemia.

A. ↑↑ Production.

1. Hemolytic disease of the newborn.
2. Hemolytic anemias $\Rightarrow$ G-6 PD deficiency - spherocytosis - Alpha-thalassemia.
3. Synthetic Vit. K.: in G-6 PD deficiency.
4. Extravasated blood $\Rightarrow$ cephalhematoma, IC hge - subcapsular hematoma of the liver.
5. Polycythemia.

B. Hepatic conjugation defect.

1. Physiological jaundice.
2. Defective conjugation. "Crigler - Najjar syndrome"
3. Inhibitors of conjugation $\Rightarrow$ Breast milk jaundice. Δ + Progesterone $\Rightarrow$ "Pec 2" β - glucuronidase
Lucey - Driscoll syndrome. "milk intolerance syndrome"
4. ↑↑ Enterohepatic circulation $\Rightarrow$
Constipation.
Pyloric stenosis.
Intestinal obstruction.
Meconium plug.
Meconium ileus.

II. Conjugated hyperbilirubinemia.

Conjugated > 20% total

A. Obstruction of bile flow.

1. Congenital biliary atresia.
2. Inspissated bile syndrome.
3. Choledocal cyst.
4. Dubin Johnson & Rotor S.

B. Hepatic cell injury.

1. Neonatal hepatitis.
2. STORCH infection.
3. Sepsis.
4. IEM $\Rightarrow$ Galactosemia.
Tyrosinemia.

Hemolytic diseases of the newborn

Erythroblastosis Fetalis

Hemolytic disease of the newborn (HDN) results from transplacental passage of maternal antibodies active against fetal RBCs $\Rightarrow$ hemolysis $\Rightarrow$ neonatal anemia & jaundice.

Rh iso-immunization

About 85% of the population are Rh+ve "DD or Dd"

While 15% are Rh-ve "dd"

- If an Rh-ve mother gets pregnant with an Rh+ve baby $\Rightarrow$ Iso-immunization is liable to occur late in pregnancy or during labour or abortion due to escape of small amount of fetal blood (Rh+ve) to maternal circulation (Rh-ve).
- The same can occur if she receives Rh+ve blood transfusion.
- Rh isoimmunization is much less frequent inspite of high rate of Rh-ve mothers $\Rightarrow$ Why?

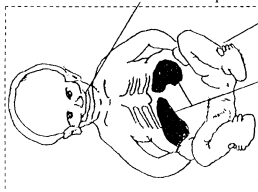
C/P

A. Hydrops Fetalis

This is the most severe form, due to severe intra-uterine hemolysis $\Rightarrow$ severe anemia & HF

1. The baby is born dead (SB) or die early after birth.

2. Marked pallor



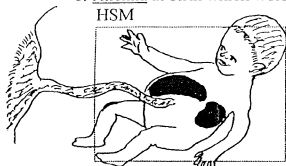
3. Severe edema, pleural effusion & ascitis.

4. Huge HSM

5. Picture of circulatory collapse weak irregular pulse, failure to start respiration.

B. Icterus Gravis Neonatorum

1. Anemia at birth which worsen rapidly during the 1st day
HSM



Jaundice $\Rightarrow$ developing during the 1st few hrs & rapidly progressive.

* If not ttt early death occurs due to kernicterus or anemic HF

- Jaundice $\Rightarrow$ 1st $\Rightarrow$ hemolytic

Later $\Rightarrow$ obstructive “ inspissated bile \$”

C. Rh – Hemolytic Anemia

- In less severe cases.
- It is usually maximum at the end of the 3rd week.
- It may necessitate packed RBCs.

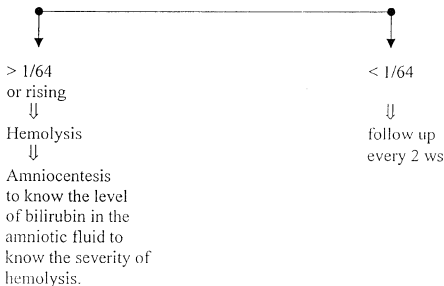
D. Very Mild Hemolysis.

- This may be detected only by lab. evidence of hemolysis.
- Otherwise the patient is normal.

Investigations

A. Ante-natal

1. Screening all females for Rh-ve persons.
2. During pregnancy
 - a. Measure maternal anti-D titre.



B. After Delivery

The cord blood is immediately analyzed for.

1. ABO & Rh group & compared to maternal blood group.
2. Hb & Hct $\Rightarrow$ if Hb < 14 = anemia.
3. S. bilirubin $\Rightarrow$ N. < 3 mg/dl
4. Retics.
5. +ve direct coomb's test.

ttt

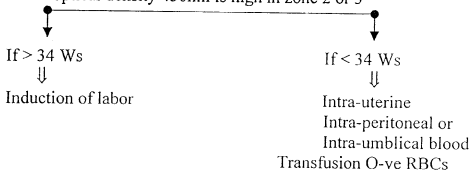
Prevention

1. Never give Rh+ve blood to Rh-ve female
2. Screening all females before marriage for Rh-blood group.
3. If Rh-ve female married to Rh+ve male
 $\Rightarrow$ Give anti-D gammaglobulin within 72hrs after birth or abortion.

Curative

A. Before birth

If spectrophotometric examination of amniotic fluid at the optical density 450nm is high in zone 2 or 3



B. After birth = ttt of hyperbilirubinemia

I. Exchange transfusion

Aims $\Rightarrow$

1. Removal of excess bilirubin
2. Correction of anemia.
3. Removal of circulating anti-D

Indications

1. S. bilirubin $\Rightarrow$
 - a. In the cord > 5mg/dl
 - b. Increasing 1mg/hr during the 1st 12 hrs
 - c. Near the critical level
- | | |
|--------------|--------------|
| 18 mg | 20mg/dl |
| $\Downarrow$ | $\Downarrow$ |
| PT | FT |

2. Cord Hb 10gm/dl or less
3. At any sign of kernicterus.
4. History of previous neonatal deaths.

Type of blood given

1. If the blood is prepared before delivery $\Rightarrow$ O-ve blood.
Fresh
Compatible with
maternal serum.
2. If prepared after delivery $\Rightarrow$ Rh-ve.
Fresh blood of the same ABO

Volume of exchanged blood.

$$= 85 \times 2 \times \text{wt (Kg)}$$

Expected effect

The serum bilirubin becomes about half the pre-exchange level.

N.B.

- A rebound increase may occur within hours after exchange which may need another exchange transfusion.

Complications

- A. Immediate $\Rightarrow$
1. Hypoglycemia.
 2. Hypothermia.
 3. Hypocalcemia.
 4. Transient bradycardia with or without Ca infusion.
 5. Cyanosis.
 6. Transient vasospasm.
 7. Apnea.

- B. Late $\Rightarrow$
1. Inspissated bile syndrome.
 2. portal vein thrombosis.
 3. Infections: CMV, HIV, hepatitis.
 4. Graft versus host disease.

N.B.

- Other indications of exchange transfusion.
 1. Neonatal septicemia.
 2. Neonatal polycythemia.
 3. Neonatal DIC
 4. Sickle cell anemia crises $\Rightarrow$ Partial exchange.
 5. Drug toxicity e.g. phenobarbitone.
 6. Metabolic diseases e.g. MSUD.

II. Phototherapy

Exposure to light especially in the blue zone $\Rightarrow$ converts the toxic unconjugated bilirubin into unconjugated isomers which can be excreted in bile & urine.

- It reduce serum bilirubin for about 1-3 mg/dl after 8-12 hrs of exposure

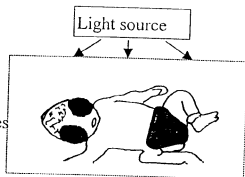
SO

- It is unsuitable to replace exchange transfusion in the critical conditions.
- Eyes & genitalia must be covered.

Complications.

4Ds

1. Dermatitis.
2. Diarrhea.
3. Dehydration.
4. Damaging effect on retina & chromosomes
5. Bronzed baby \$ if applied to infant with direct hyperbilirubinemia.



III. Phenobarbitone.

$\Rightarrow \oplus$ Glucuronyl transferase enzyme.

Dose $\Rightarrow$ 3-8 mg /Kg/D even up to 10mg /Kg/D

IV. Packed RBCs

May be needed if anemia persist after disappearance of jaundice.

V. Hypoglycemia.

Commonly affects the newborn with Rh-incompatibility due to islet cell hyperplasia.

ttt $\Rightarrow$ see hypoglycemia.

ABO-Incompatibility

Although ABO incompatibility occurs in 25% of pregnancies $\Rightarrow$ hemolytic disease occurs in only less than 10% of such offspring due to

1. Low antigenicity of fetal ABO factors.
2. Maternal Abs are of the IgM $\Rightarrow$ doesn't cross the placenta

Character

1. The mother $\Rightarrow$ group O
Baby $\Rightarrow$ group A or B.
2. The 1st baby can be affected.
3. Onset of jaundice during the 1st 24 hrs but not at birth.
4. Mild course, mild anemia, mild HSM & less incidence of kernicterus.
5. Coombs test - ve or weak + ve.
6. Lab. findings.
 - Mild anemia.
 - Anti-A or anti-B can be detected in the baby's serum.
7. Photo-therapy may be enough in mild cases but exchange transfusion may be needed in severe cases.

Physiological Jaundice

- The most common.
- It affects 50% FT & more common & more severe in preterm "PT"

Etiology

1. Immature glucuronyl transferase enz.
2. Immature Z & Y-proteins "ligandin"
3. Short life span of RBCs $\Rightarrow$ $\uparrow\uparrow$ rate of destruction.

It's induced by physiological factors

Character

1. Onset $\Rightarrow$ 2nd - 3rd day.
2. Disappears before 7th day.
3. In PT $\Rightarrow$ Earlier onset.
Reach higher peak.
Disappears before 14th D.
4. S. bilirubin doesn't rise over 10 mg/dl in FT & 14 mg/dl in PT if is of unconjugated type.

Important tests:
No pale stool
No hematuria
No dark urine
No jaundice
No anemia
No hepatomegaly
No splenomegaly
No fever
No weight loss
No dehydration
No acidosis
No hypocalcemia
No hypoglycemia
No hypothermia
No hypoxia
No hypernatremia
No hyponatremia
No hyperkalemia
No hypokalemia
No hypermagnesemia
No hypomagnesemia
No hyperphosphatemia
No hypophosphatemia
No hypercalcemia
No hypocalcemia
No hypernatremia
No hyponatremia
No hyperkalemia
No hypokalemia
No hypermagnesemia
No hypomagnesemia
No hyperphosphatemia
No hypophosphatemia
No hypercalcemia
No hypocalcemia

ttt

1. No ttt in mild cases.
2. Phototherapy & phenobarbitone are useful to prevent dangerous rise of unconjugated bilirubin.

It's the most God's important gift

Criggler - Najjar Syndrome

Due to congenital absence of glucuronyl transferase enzyme.

Type I (AR)

1. Complete absence
↓
2. Severe jaundice
↓
3. Kernicterus
↓
4. Early death

Type II (AD)

1. Partial deficiency
↓
2. Mild jaundice
↓
3. Good response to phenobarbitone

Breast Milk Jaundice

Progesterone $\Rightarrow$ 3α 20 β - pregnandiol $\Rightarrow$ secreted in milk $\Rightarrow$

⊖ Glucuronyl transferase enz.

ttt

Stop breast feeding for only 48 hrs $\Rightarrow$ then continue it again.

N.B. In Lucy Driscoll's the inhibiting substance is not identified.



Happy Mother's Day

It's motherhood ...

Cong. biliary atresia.

The most common cause of obstructive jaundice in the neonatal period.

Intra-hepatic

Extra-hepatic

C/P

1. Onset $\Rightarrow$ 2nd week.
2. Course $\Rightarrow$ progressive
3. Obstructive type
 - a. Urine $\Rightarrow$ dark
 - b. Stool $\Rightarrow$ pale "clay colored"
 - c. Olive green discoloration of skin & sclera.
4. Progressive hepatomegaly $\Rightarrow$ deterioration of liver functions.
5. Portal hypertension, ascitis, bleeding tendency & death due to liver cell failure.

Investigations

See neonatal hepatitis.

ttt

1. Extra-hepatic $\Rightarrow$ surgical
2. Medical care of the baby nutrition
 - a. Medium chain triglyceride instead of fat.
 - b. Fat soluble vitamins should be given by injection
"Vit. A,D, K, E"
3. Management of liver cirrhosis & liver cell failure.

Neonatal Hepatitis

Etiology

1. Idiopathic
2. Infections
 - a. Viruses $\Rightarrow$ Hepatitis B or C
CMV
Herpes simplex
EBV
Rubella

- b. Bacteria ⇒ Septicemia
 Portal pyemia.
 Cong syphilis.
- c. Protozoa ⇒ Toxoplasmosis

C/P

Very similar to biliary atresia

Investigations

See the following table .

ttt

Bacterial ⇒ Antibiotics according to culture & sensitivity.

Viral ⇒ Acyclovir, interferon.

Toxoplasmosis ⇒ Daraprim or tripple sulpha.

<i>Investigations</i>	<i>Biliary Atresia</i>	<i>Neonatal hepatitis</i>
S. Bilirubin SGOT, SGPT Alk phosphatase..	↑↑↑ Direct ↑ Mild ↑↑↑ Marked.	↑↑↑ Biphasic ↑↑↑ Marked ↑ Mild.
U/S	1. Extrahepatic ⇒ dilated intrahepatic ducts 2. Intrahepatic ⇒ absent bile ducts.	Both intra& extrahepatic bile ducts are present.
HIDA scan	*Rapid hepatic uptake *Absent excretion into intestine..	• Poor uptake by the liver cells • Excretion into the intestine occurs
Liver biopsy	As U/S	• Infiltration by inflammatory cells + distortion of lobular architecture. • Bile ducts are present..

It's time to work hard

Kernicterus

Definition

Yellowish staining of certain brain nuclei especially the basal ganglia in the newborn as a result of high serum indirect bilirubin
⇒ neuronal necrosis ⇒ replaced by glial cells.

Etiology

- Neonatal indirect hyperbilirubinemia due to any cause may lead to kern-icterus.
 - Serum bilirubin should rise above certain level.
 - This level is determined by the maturity & vitality of the blood brain barrier "BBB"
 - In FT = BBB is immature so the level is lower.
 - Certain conditions further increase the incidence of kernicterus due to,
 1. ↑↑ susceptibility of CNS to the toxic effect of bilirubin.
 2. Affects the BBB
 3. Compete with bilirubin for its albumin binding sites.
- e.g.
- a. Hypoglycemia.
 - b. Hypoxia.
 - c. Acidosis.
 - d. Hypothermia.
 - e. Sepsis & sulfa.

C/P

Stage I

Poor Moro reflex.
Hypotonia
Lethargy
Poor feeding
Vomiting.
High pitched cry.

Stage II

Opisthotonus
Convulsions.
Rigidity.
Oculogyric crises.

Stage III.

The rigidity disappears & the general condition improves.

Stage IV

C P, deafness, choreoathetosis, MR.

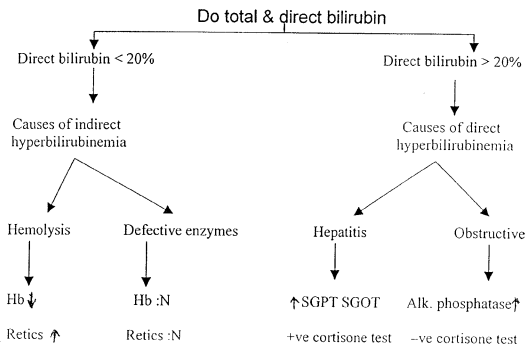
Investigations

Of neonatal jaundice.

ttt

- Prevention & ttt of indirect hyperbilirubinemia.
- Prevention & proper management of factors which $\uparrow\uparrow$ bilirubin toxicity.

How to investigate a case of neonatal hyperbilirubinemia



Hemorrhagic Disease of the newborn

It is spontaneous bleeding tendency affecting the newborns & preterms from the 2nd to 6th day of life.

Etiology

1. Vit. K deficiency $\Rightarrow$ due to absence of intestinal flora at birth.
2. Maternal deficiency of Vit. K.
3. Immaturity of the liver $\Rightarrow$ $\downarrow$ clotting Factors
4. Increased capillary fragility (in preterms).

Sites of hge.

GIT, nose, umbilical stump intracranial, pulmonary.

Investigations

1. $\uparrow$ PT
2. BT $\Rightarrow$ N.
3. PTT $\Rightarrow$ N
4. $\downarrow$ Factors II, VII, IX, X.
5. CBC $\Rightarrow$ normal platelet count.

Prevention

1. Give the mother $\Rightarrow$ 5-10 mg Vit K, IM 4-6 hrs before delivery.
2. Give the newborn $\Rightarrow$ 1mg Vit K1, IM immediately after birth.

ttt

1. Vit. K1 $\Rightarrow$ 1-5 mg IM or IV
2. Blood transfusion (Fresh whole blood) or FFP $\Rightarrow$ in severe cases.

D.D. of hge in the newborn

A. Hemophilias, A, B, C & Von Willibrand disease.

$\Rightarrow$ See hematology.

B. DIC : usually as a complication of sepsis, anoxia or acidosis.

⇒ See hematology.

C. Thrombocytopenia :

1. Isoimmune thrombocytopenia.
2. Maternal ITP.
3. Maternal SLE.
4. TAR S.
5. STORCII infection.
6. Neonatal sepsis.

C. Swallowed blood :

The infant may swallow the mother's blood during delivery or from a fissure in her nipple. Later blood is passed in the stool or is vomited.

In this condition alkaline denaturation test is essential to differentiate maternal blood from fetal blood.

Clinical evaluation	Laboratory studies			Diagnosis
	Platelets	PT	PTT	
<i>Sick</i>	↓	↑	↑	DIC
	↓	N	N	Thrombocytopenia
	N	↑	↑	Liver disease
	N	N	N	Vascular causes as in hypoxia , acidosis & premature
<i>Healthy</i>	↓	N	N	Immune thrombocytopenia & BM hypoplasia
	N	↑	N	Hemorrhagic diseases of the newborn
	N	N	↑	Hemophilia
	N	N	N	Bleeding due to local factor

Cyanosis in the Newborn

Etiology

A. Respiratory distress

Peripheral

1. RDS
2. Aspiration of amniotic fluid
3. Pneumonia
3. Atelectasis

Central

1. IC hge
2. Narcotics
3. Perinatal anoxia

B. Congenital cyanotic HD

1. TGA
2. Single ventricle
3. "Truncus arteriosus
4. Pulmonary atresia.
5. Severe F4

C. Methemoglobinemia.

D. Metabolic

- Hypoglycemia.
- Septicemia.
- Hypothermia.

Anemia in the newborn

It is usually physiological or pathological caused by :

1. Hemolysis
2. Hemorrhage.
3. BM depression.

Etiology

A. physiological anemia of the newborn:

1. Physiologic anemia of infancy:

After birth , there is rapid fall in Hb level , starting by the end of the 1st week of life and progressively declining reaching a lowest level at 6-8 weeks after birth . this a physiological phenomena called physiologic anemia of infancy and this is due to :

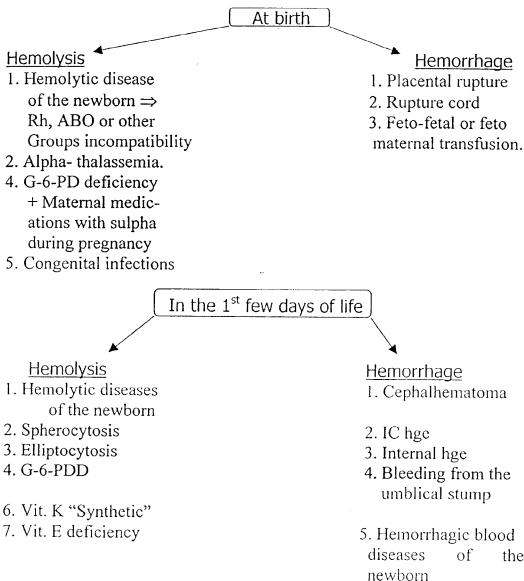
1. Shorter life span of the RBCs containing the fetal Hb.
2. Abrupt cessation of erythropoietin at birth as a result of onset of respiration and increased O₂ saturation.

Once the lowest level is reached , RBCs production is stimulated with gradual increase in Hb .

3. Anemia of prematurity:

It is an exaggeration of the normal physiological anemia . In addition , small premature infants develop rapidly vit. E deficiency unless supplied exogenously. Such vit. E deficiency causes more shortening of the life span of RBCs.

B. Pathological anemias in the neonatal period



Iatrogenic

Repeated blood sample

Investigations

1. CBCs & reticulocytic count.
2. Coomb's test.
3. G-6-PD assay.

4. Osmotic fragility test.
5. STORCH-EB screening.

ttt

1. ttt of underlying cause.
2. Packed RBCs transfusion if Hb < 10 gm%.
3. Recombinant human erythropoietin .

Fever in the newborn

- 2 important causes of fever in the neonate $\Rightarrow$ Dehydration fever
Neonatal infections

Dehydration fever

The most common cause of fever in the neonatal period

Etiology

1. Delayed feeding due to delayed breast milk production without fluid supplementation.
2. Exposure to high environmental temp.
3. Over-clothing especially in hot weather.

C/P

1. High body temp. (may be 40)
2. Hot dry, flushed skin.
3. *C/P of dehydration* depressed anterior fontanel, sunken eyes dry mucus membranes, dry tongue thirst & oliguria.
4. Convulsions may occur due to?

ttt

1. Correction of dehydration.
2. The condition is better prevented by early feeding of the baby.
3. Fluid supplementation is required esp. in hot weather.

Neonatal Infections

The infections of the newborn are discussed later in the notebook of infection.

Maternal diseases affecting the newborn

A. Infections

See neonatal infections .

B. Non-infectious diseases

1. Maternal Diabetes

- * Prematurity
- * RDS
- * neonatal hypoglycemia.
- * Macrosomia $\Rightarrow$ birth trauma

2. Toxemia, hypertension, renal disease.

- $\Rightarrow$
- * IURG $\Rightarrow$ small for date.
 - * Prematurity.
 - * IUFD

3. Autoimmune disease mediated by Ig G: SLE, myasthenia gravis. ITP & grave's disease.

4. Maternal PKU

- * Microcephaly
- * MR
- * Abortion
- * Small for date.

5. Osteomalacia.

- * Hypocalcemia.
- * Rickets.

Nonatal Convulsion

Etiology

1. Cerebral malformations, trauma, hge or anoxia.
2. Infections.
 - * Cerebral $\Rightarrow$ meningitis, encephalitis.
 - * Septicemia.
 - * Tetanus neonatorum.
3. Metabolic.
 - * Hypoglycemia.

- * Hypocalcemia.
 - * Hypomagnesemia.
 - * Hyper-natremia.
 - * Vit. B6 deficiency.
 - * IEM $\Rightarrow$ Galactosemia, MSUD
4. Toxins.
- * Drugs $\Rightarrow$ minophylline, coramine.
 - * Metabolic toxins.
 - Bilirubin $\Rightarrow$ kernicterus.
 - Uremic toxins.

Investigations.

To reach the cause

1. CBC + CRP $\Rightarrow$ for infections.
2. CSF examination.
3. Brain sonar.
4. Electrolytes + bilirubin + renal function.

ttt

1. Assurance of patent airway + O₂
2. Control of convulsions by:
 - a. Phenobarbitone.
 - 5mg/ kg IM
 - Can be repeated after 30 min.
 - b. Diazepam " valium"
 - 0.1 - 0.3 mg/kg IV
3. TTT of the cause.



Now .. We're the best

Edema in the Newborn

A. Generalized

1. Severe hemolytic disease of the newborn.
2. HF $\Rightarrow$ due to congenital HD
3. Congenital nephrotic syndrome.

B. Localized

1. Caput succedaneum.
2. Cord around the neck.
3. Congenital lymphedema.
4. Turner syndrome.



Till next time ...

Routine Care of The Neonate at Birth and Neonatal Resuscitation

- 1) The newly born baby is gently dried and wrapped in a warm, sterile towel.
- 2) The time of birth is noted.
- 3) Put the baby on a radiant warmer on his back with the neck in the neutral position (neither flexed nor extended to avoid airway compression) by putting a towel beneath the baby's shoulders.

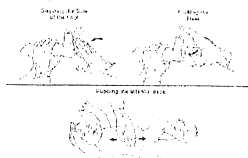


- 4) The upper airway is cleared by gentle suction of excess fluid and any inhaled vernix, blood or meconium: start by suctioning the mouth and pharynx since nasal stimulation by the suction catheter can induce a strong gasp with aspiration of oral and pharyngeal contents. Avoid too deep suctioning of the pharynx since this will induce vagal stimulation and bradycardia.
- 5) If the baby starts to breathe regularly with his color changing to pink no further resuscitative steps are needed. If not look at step (7).
- 6) Complete routine care by:
 - The umbilical cord is clamped and cut with sterile blades and the stump is cleaned with 70% alcohol and the entire skin is cleaned with sterile cotton soaked in mild soap solution.
 - Vitamin K: 1 mg IM is given to avoid hemorrhagic disease of the newborn
 - The baby is weighed and rapid clinical examination is done.
 - Care of the eyes is done by application of silver nitrate 1% or chloramphenicol eye drops.
 - The baby is dressed and put in the arms of his mother. The latter should be encouraged to put her baby as early as possible on her breast so as to enhance maternal neonatal bonding by early skin-to-skin contact.
- 7) If the baby is taking regular breaths but his color remains cyanotic he is in



need for additional oxygen by facemask till color improves.

- 8) If the baby fails in taking regular breaths or remains apneic do
 a) Gentle stimulation by rubbing the back or slapping the soles of the feet.



- b) In many cases blowing oxygen on the nose will initiate breathing.
 c) If all of these steps fail the next step is to:
 i- Expand and ventilate the lungs with either bag and mask or bag and endotracheal tube. Often inflation of the lungs itself will initiate a gasp with spontaneous breathing. If it does not, the lungs should be ventilated at a rate between 30 and 50 inflations per minute with pressures below 30 cm H₂O. The majority of infants will quickly become pink and begin breathing within 2-5 minutes.



- ii- If the infant fails to become pink with good expansion of the lung then he is probably in a state of shock. Cardiac massage using two-finger pressure on the lower half of the sternum at a rate of 140 taps per minute is occasionally necessary.



- iii- Correction of acidosis by alkali injection, and persisting bradycardia by adrenaline administration into the umbilical vein can be helpful. If no improvement rapid intravascular expansion is done.

To summarize:

- Resuscitation is an orderly process which is done step by step whenever a neonate is in need for it depending on the color, breathing pattern and heart rate of the baby.
- Resuscitation steps are summarized by the letters ABCDE
 - A $\Rightarrow$ Airway which should be suctioned, cleared and opened by proper positioning of the head.
 - B $\Rightarrow$ Breathing which is stimulated by
 - Tactile stimulation
 - Blowing O₂ in the nose
 - Bag and mask ventilation
 - Ventilation by bag and endotracheal tube
 - C $\Rightarrow$ Circulatory support by cardiac massage in case of persistence of cyanosis and/or bradycardia in spite of proper chest expansion.
 - D $\Rightarrow$ Drugs used in resuscitation include:
 - Adrenaline (1/10.000 solution) 0.2 ml / Kg give either IV or via endotracheal tube and can be repeated
 - Sodium bicarbonate (8.4%) 2 ml / Kg = 2 mEq/Kg diluted 1:1 in glucose 5%.
 - Naloxone (Narcan) 0.1 mg/Kg/dose give IV, IM or via endotracheal tube if opiate narcotics were given to the mother in the past 4 hours.
 - Volume expanders given in case of shock as 20 ml/Kg of normal saline 0.9% or Ringer's solution or 10 ml/Kg of packed RBCs (in case of severe hydrops with heart failure).
 - E $\Rightarrow$ Evaluation of the success of the step or maneuver done in the resuscitation.
- APGAR score (named so according to Dr. Virginia Apgar, an American anesthetist) is a practical method which was used before for systematically assessing the newborn infant immediately after birth, at the 1st minute of life and thereafter depending on 5 objective signs (see table), but waiting till 1 minute to start inflation of the lungs whenever needed represent a lengthy delay in a life saving maneuver.

NEONATAL SCREENING

Definition:

Screening can be defined as the search for occult disease or the potential for disease to develop, in a defined population.

Aim:

The purpose is primarily to screen all newborn infants for a given disorder in which symptoms would not be clinically present until irreversible damage occurred and for which an effective treatment is available.

Timing:

Neonatal screening tests include a group of assays, which are carried out between the 3rd and 6th days of life, and become an essential measure for early detection of some inborn genetic defects.

Screening tests characteristics:

- The disease should be serious and common enough to warrant the effort and mass screening
- Early detection of the disease should be beneficial in terms of ease of treatment and improved prognosis
- The neonate should be normal at birth
- Follow-up mechanisms must be available.
- The screening tests must be easy to perform and interpret, and have acceptably low risk morbidity and cost with high sensitivity and specificity.

Neonatal screening for various disorders:

1- Clinical neonatal screening

- Congenital heart disease
- Congenital dislocation of the hip
- Tracheo-oesophageal fistula
- Imperforate anus

2- Neonatal screening for inborn errors of metabolism

Some examples are listed in the following table.

Disorder	Frequency	Mode of inheritance	Clinical picture
Phenylketonuria	1:14000	AR	Irreversible MR
Galactosemia	1:60000	AR	Vomiting, diarrhea, jaundice and failure to thrive, cataract in early infancy
Maple syrup urine disease	1:200000	AR	Severe acidosis, hypoglycemia, seizures, maple syrup odor
Homocystinuria	1:200000	AR	Marfanoid appearance
Hereditary fructose intolerance	1:20000	AR	Acute hypoglycemia, chronic hepatic & renal dysfunction

3- Neonatal screening for endocrine disorders

a- Congenital hypothyroidism

- The incidence has been estimated to be 1 in 4000 births
- Routine newborn screening in USA, Canada, most western countries and recently established in Egypt
- Carried out in dried spot samples collected via skin puncture obtained at 3-5 days after birth.
- T4 is measured initially, about 34% of low T4 values detected are not the result of true hypothyroidism, but represent diminished levels of thyroxin binding globulin (TBG).
- If the initial T4 is low, repeat T4 and TSH on the same sample with radioimmunoassay or with delayed fluorescence-immunoassay.
- If the repeated T4 is still low ($< 6 \text{ microgm / dl}$) or the TSH is elevated, confirmatory serum sample should be obtained.
- Newborn infants with concurrent illness or congenital malformation may have transient TSH elevation. So re-evaluation of the diagnosis of congenital hypothyroidism in such infants is indicated.

b- Congenital adrenal hyperplasia

- The incidence of the classic form of this disorder varies from 1: 5000 to 1: 20000 depending in the population.
- Deficiency of 21 hydroxylase activity accounts or more than 90% of cases.
- Clinical diagnosis of 21-OH-D-CAH is poor in the neonatal period as clinical features range from classic salt-losing virilizing form to simple virilizing picture characterized by premature pubarche.

4- Neonatal screening for blood diseases

a- Screening for hemoglobinopathies

- i. Sickle cell disease.
- ii. Hemoglobin e
- iii. Hemoglobin F

b- Screening for G6PD deficiency

5- Other disorders considered for neonatal screening

- a- Cystic fibrosis
- b- Alpha-1 antitrypsin deficiency
- c- Duchenne muscular dystrophy
- d- Neuroblastoma
- e- Familial ureteric reflux
- f- Liver diseases and biliary atresia
- g- Neonatal hearing screening
- h- Screening for congenital infections

Hypoxic – Ischemic Encephalopathy

Etiology

A. Fetal hypoxia

1. Maternal hypoventilation

- During anesthesia
- HF
- Carbon monoxide poisoning

2. Maternal hypotension

- Spinal anesthesia
- Compression of IVC & Aorta by the uterus
- Dehydration

3. Compression of umbilical cord impeding circulation of blood.

4. Uterine causes

- uterine tetany $\Rightarrow$ due to excessive oxytocin dose

5. Placental causes

- Premature separation
- Placental insufficiency.

B. Neonatal hypoxia

1. Severe anemia: Hemolysis hge

2. Shock: Adrenal hge IC hge Infection

3. CNS depression: Narcotics Injury

4. Pulmonary: RDS

5. Cong. Cyanotic heart disease.

C/P

1. IUGR due to fetal hypoxia
2. fetal heart rate slows & beat to beat variability declines.
3. fetal scalp blood analysis $PH < 7.2$
4. Meconium stained AF = fetal distress
5. At birth $\Rightarrow$ there's marked CNS depression with failure of spontaneous respiration
6. During resuscitation there's severe hypotonia which may change rapidly to hypertonia or normal tone.
7. Pallor, Cyanosis, apnea & slow HR.
8. Cerebral edema develops during the 1st 24hrs. $\Rightarrow$ severe brain stem depression.
9. Convulsions may occur & may be resistant to anticonvulsant therapy.
 $\rightarrow$ causes of convulsions are?
10. Disturbed level of consciousness.

Management

1. Avoidance of the causes of possible.
2. Monitoring of fetal wellbeing during pregnancy & labor.
3. Resuscitation
4. ttt of meconium aspiration .

Post-natal management

Investigations

- | | |
|--------------|-------------------------|
| 1. EEG | 5- ABG |
| 2. Brain U/S | 6- Renal function tests |

3. CT scan.
4. Creatine Kinase.

Treatment

1. O_2 level should be corrected by O_2 therapy.
2. Dobutamine $\Rightarrow$ to improve perfusion.
3. Correction of associated metabolic disorders $\Rightarrow$
hypoglycemia
Hypocalcemia
hypomagnesemia
4. Control of convulsions.

Prognosis

1. Death 10-20 % of infants.
2. C.P 20-45%
3. No brain damage 30-35%.

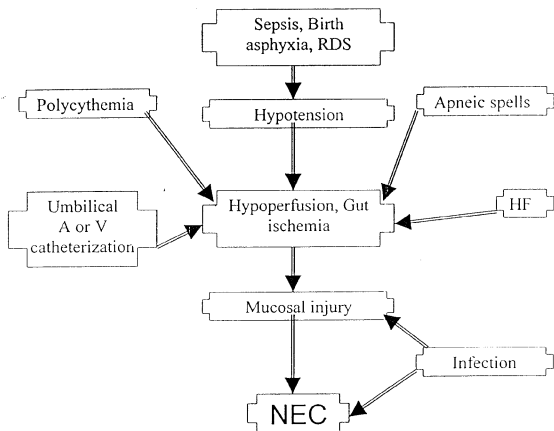


Please... give me it

Necrotizing Enterocolitis (NEC)

The disease commonly affects the ileum & colon but no part of the GIT is excluded

Etiology & Pathogenesis



C/P

NEC should be expected as a very probable complication in P.T. especially if there's predisposing factors.

I. Abdominal manifestations:

1. Abdominal distension.
2. Delayed gastric emptying.
3. vomiting which may be bilious.
4. bleeding/rectum & hematemesis may occur.
5. O/E → marked abdominal tenderness.

II. Systemic manifestations

1. Respiratory distress, Apnea.
2. Temperature instability.
3. Hypotension & shock
4. Bleeding tendency.

Investigations

1. CBC: ⇒ Thrombocytopenia
2. Blood gases ⇒ persistent metabolic acidosis.
3. Refractory hyponatremia 1,2 & 3 are important triad in NEC.
4. Stool ⇒ blood in stool.
5. Plain X-ray erect abdomen ⇒ Pneumo-peritoneum

Management

- No oral feeding
- Nasogastric tube suctioning
- Begin parenteral feeding through peripheral vein.
- Treat any predisposing cause or complications as →

1. Infections

According to culture & sensitivity if the result not obtained start with penicillin -Garamycin - flagyl IVI.

Continue antibiotics for at least 14 days.

2. GIT

Pediatric surgeon must be consulted if there's perforation.

3. Cardio-respiratory

- Regular follow up by blood gases patient may need assisted ventilation.
- Plasma expanders or fresh blood or F.F.P.

4. Metabolic

NaHCO_3 to correct metabolic acidosis

5. Blood

Platelet conc. $\Rightarrow$ thrombocytopenia

Exchange transfusion may be needed.

